=> fl caplus

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=> s mutat? and superantigen#

	170114	MUTAT?
	2307	SUPERANTIGEN#
L1	107	MUTAT? AND SUPERANTIGEN#

=> s streptoco? and l1

	29456	STREPTOCO?
L2	6	STREPTOCO? AND L1

=> d l2 1-6 bib ab

L2 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2000 ACS
AN 1998:649642 CAPLUS
DN 130:13058
TI **Mutational** analysis of **superantigen** activity responsible for the induction of skin erythema by **streptococcal** pyrogenic exotoxin C
AU Yamaoka, Junichi; Nakamura, Eijiro; Takeda, Yoshifumi; Imamura, Sadao; Minato, Nagahiro
CS Department of Dermatology, Graduate School of Medicine, Kyoto University, Kyoto, 606-8501, Japan
SO Infect. Immun. (1998), 66(10), 5020-5026
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English

AB **Streptococcal** pyrogenic exotoxin C (SPEC), when injected intradermally, induces erythema in unsensitized rabbits. In the present study, we examd. whether this erythema induction is due to the T-cell stimulatory activity of SPEC as a **superantigen**. Anal. by using single-residue mutant SPECs indicated that mutant SPECs Y15I, A16E, and Y17I, in which tyrosine 15, alanine 16, and tyrosine 17 were replaced with isoleucine, glutamic acid, and isoleucine, resp., exhibited significantly reduced mitogenic activity for V.beta.2+ human T cells in vitro, and Y15I showed as much as a 1,000-fold redn. Y15I mutant SPEC, however, retained the ability to bind to major histocompatibility complex class II antigen and to form a homodimer, implying that residue 15 is critically important for the interaction of SPEC with T-cell antigen receptor .beta. chains. When injected intradermally into normal rabbits, wild-type SPEC induced a characteristic erythema after 3 h in a dose-dependent fashion, which was assocd. with polymorphonuclear and mononuclear cell infiltration. This erythema formation was found to be severely suppressed by systemic pretreatment with cyclosporin A, suggesting the involvement of host T cells. Y15I mutant SPEC exhibited nearly 1,000-fold less erythema induction in vivo than wild-type SPEC. Altogether, the present results strongly suggest that erythema induction in rabbits by SPEC is attributable mostly to its T-cell stimulatory activity as a **superantigen**.

L2 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2000 ACS
AN 1997:408618 CAPLUS
DN 127:46293
TI Analysis of toxicity of **streptococcal** pyrogenic exotoxin A mutants
AU Roggiani, Manuela; Stoehr, Jennifer A.; Leonard, Bettina A. B.; Schlievert, Patrick M.
CS Department of Microbiology, University of Minnesota, Minneapolis, MN, 55455, USA
SO Infect. Immun. (1997), 65(7), 2868-2875
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
AB In this work we generated single- and double-site **mutations** of **streptococcal** pyrogenic exotoxin A (SPE A) at residues K16, N20, C87, C90, C98, K157, S195, N20/C98, and N20/K157. The mutant SPE A's were analyzed in vivo for their lethal activity and in vitro for their superantigenic ability. Our results indicate that SPE A's ability to induce lethality and endotoxin enhancement does not require superantigenicity, and conversely superantigenicity does not necessarily lead to lethality. Thus, these properties and their relative contributions to the onset of hypotension and shock may be separable. Furthermore, evidence is presented that certain mutant toxins may be suitable for use as vaccine toxoids.

L2 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2000 ACS
AN 1997:272238 CAPLUS
DN 126:329221
TI Analysis of the interaction between the bacterial **superantigen** **streptococcal** pyrogenic exotoxin A (SpeA) and the human T-cell receptor
AU Kline, J. Bradford; Collins, Carleen M.
CS Department of Microbiology and Immunology, University of Miami School of Medicine, Miami, FL, 33101, USA
SO Mol. Microbiol. (1997), 24(1), 191-202
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell
DT Journal
LA English

AB **Streptococcus** pyogenes that produces the bacterial **superantigen streptococcal** pyrogenic exotoxin A (SpeA) is assocd. with outbreaks of **streptococcal** toxic shock syndrome (STSS) in the United States and Europe. SpeA stimulates V.beta.2.1, 12.2, 14.1, and 15.1-pos. T cells, and the lymphokine prodn. from the activated T cells is believed to result in the symptoms assocd. with STSS. The T-cell receptor (TCR)-SpeA interaction is crucial for superantigenic activity, and studies were undertaken to det. regions of both SpeA and the TCR involved in the formation of MHC/SpeA/TCR complexes. Previously, recombinant toxins encoded by speA alleles 1, 2, and 3 as well as toxins resulting from 19 distinct point **mutations** in speA1 were generated. Here, these 22 toxin forms were incubated with human peripheral blood mononuclear cells (PBMCs), and the percentages of T-cell blasts bearing V.beta. chains 2.1, 12.2, and 14.1 were quantified by flow cytometry. The anal. indicates that the residues of SpeA needed for a productive TCR interaction differ for each V.beta. chain examd. An amino acid substitution at only one site significantly affected the toxin's ability to stimulate V.beta.2.1-expressing T cells, three individual amino acid substitutions resulted in significant loss of ability to stimulate V.beta.12.2-expressing T cells, and substitution at 13 individual sites significantly affected the ability to stimulate V.beta.14.1-expressing T cells. To elucidate the regions of the V.beta. chains that interacted with SpeA, synthetic peptides representative of the human V.beta.12.2 complementary-detg. regions (CDRs) 1, 2, and 4 were used to block the SpeA-mediated proliferation of human PBMCs. The CDR1, CDR2, and CDR4 peptides were each able to block proliferation with the activity of CDR1>CDR2>CDR4. Combinations of CDR1 peptide with CDR2 or CDR4 peptides allosterically enhanced the ability of each to block proliferation, suggesting SpeA has distinct binding sites for the CDR loops.

L2 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2000 ACS
AN 1996:725553 CAPLUS
DN 126:17531
TI Altered orientation of **streptococcal superantigen** (SSA) on HLA-DR1 allows unconventional regions to contribute to SSA V.beta. specificity
AU Stevens, Kristin Reda; Van, Mai; Lamphear, James G.; Rich, Robert R.
CS Dep. of Microbiology, Baylor College of Medicine, Houston, TX, 77030, USA
SO J. Immunol. (1996), 157(11), 4970-4978
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB Crystallog. studies reveal even distantly related bacterial **superantigens** (SAg) to adopt a common structural topol. **Mutational** analyses confirm that this shared folding pattern often confers a conserved function to analogous residues in different SAg, albeit with specificities for particular TCR or MHC class II mols. It was thus surprising that the **streptococcal** SAg SSA differed from related SAg in the location of its V.beta.-detg. residues. Because it seemed unlikely that SSA would deviate significantly from an SAg-like topol., we hypothesized that variations in SSA V.beta.-detg. regions might result from differences in SSA-MHC class II interactions relative to other SAg during SSA presentation to the TCR. Comparison of the DR1-binding properties of SSA with its closest homolog SEB found different amino acid positions within SAg primary sequences to contribute to SSA-DR1 and SEB-DR1 interactions, and suggested that SSA bound DR1 with an altered orientation relative to SEB. The common involvement of DR1 .alpha.39K, however, predicted that the two SAg bound overlapping sites on DR1.

Nevertheless, SSA and SEB did not effectively cross-compete for DR1 binding and had opposite patterns of DR1-binding affinity in the presence of distinct DR1-expressing cell lines. The data thus suggest that SSA and SEB bind not only with different orientations on DR1, but may bind preferentially, to distinct DR1 subsets delineated by cell-specific factors. Differences in orientation of SSA on DR1 and/or interaction of SSA with particular DR1 subsets delineated by cell-specific factors. Differences in orientation of SSA on DR1 and/or interaction of SSA with particular DR1 subsets may explain why unconventional regions influence SSA TCR V.beta. specificity.

L2 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2000 ACS
AN 1996:480428 CAPLUS
DN 125:137692
TI Structure-function analysis of the **superantigen** staphylococcal enterotoxin C1 by mutagenesis (Staphylococcus aureus, **Streptococcus** pyogenes, toxic shock)
AU Hoffmann, Marcy Lynn
CS Univ. of Idaho, Moscow, ID, USA
SO (1995) 178 pp. Avail.: Univ. Microfilms Int., Order No. DA9621792 From: Diss. Abstr. Int., B 1996, 57(3), 1602
DT Dissertation
LA English
AB Unavailable

L2 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2000 ACS
AN 1993:647528 CAPLUS
DN 119:247528
TI **Mutations** affecting MHC class II binding of the **superantigen streptococcal** erythrogenic toxin A
AU Hartwig, Udo F.; Fleischer, Bernhard
CS 1st Dep. Med., Univ. Mainz, Mainz, D-6500, Germany
SO Int. Immunol. (1993), 5(8), 869-75
CODEN: INIMEN; ISSN: 0953-8178
DT Journal
LA English
AB **Streptococcal** pyrogenic exotoxin A (SPEA) is an important pathogenicity factor of group A **streptococci**. It is a member of the family of **superantigens** produced by Staphylococcus aureus and **Streptococcus** pyogenes, and its T lymphocyte stimulating activity is involved in the pathogenesis of certain diseases caused by pyogenic **streptococci**. In this study the authors have generated 9 mutant SPEA mols. by substituting amino acids in the regions of homol. between different **streptococcal** and staphylococcal **superantigens**. An addnl. mutant was created by deletion of the 10 N-terminal amino acids. The mutants were expressed as fusion proteins. Several **mutations** led to a loss of function due to a loss of class II-binding activity. Such loss **mutations** did not cluster to a certain region of the SPEA mol. Rather, even a substitution of neighboring amino acids had opposite effects. None of the loss **mutations** affected the binding of neutralizing mAb and all loss mutants could be pptd. in Ouchterlony tests by a polyclonal anti-SPEA serum. Thus, the functional activities of SPEA, and probably of other **superantigens** as well, cannot be attributed to a defined region of the mol.

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=> d his

(FILE 'HOME' ENTERED AT 16:12:20 ON 04 JAN 2000)

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     FILE 'CAPLUS' ENTERED AT 16:13:22 ON 04 JAN 2000
L1          107 S MUTA[redacted]AND SUPERANTIGEN#
L2            6 S STREPTOCO? AND L1

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CA SUBSCRIBER PRICE                                    -2.78      -2.78

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MUTATA	1
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MUTATABLE	25
MUTATABLE13	1
MUTATABLY	2
MUTATAGEN	1
MUTATAGENESIS	1
MUTATAGEN-LINKED-THIRD	1
(MUTAT$ AND STREPTOCO$ AND TOXIN AND SHOCK).ALL.	94

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